I.

A CLINICAL CONTRIBUTION

TO THE STUDY OF

POST-PARALYTIC CHOREA

II.

A CONTRIBUTION

TO THE STUDY OF

LOCALIZED CEREBRAL LESIONS

BY

E. C. SEGUIN, M.D.

CLINICAL PROFESSOR OF DISEASES OF THE MIND AND NERVOUS SYSTEM, IN THE COLLEGE OF PHYSICIANS AND SURGEONS, NEW YORK; PRESIDENT OF THE NEW YORK NEUROLOGICAL SOCIETY.

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G. P. PUTNAM'S SONS
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A CLINICAL CONTRIBUTION

TO THE

STUDY OF POST-PARALYTIC CHOREA.

By E. C. SEGUIN, M.D., of New York.

Having had the opportunity of observing two well-marked instances of this remarkable symptom-group, and as I am able to bring forward one of the patients, I make bold to call the attention of the Association to the subject. Before reciting the cases, it may not be amiss to say a few words of the growth of our knowledge of the subject. Although the symptom-group had been observed a number of years ago by various physicians, yet it was our distinguished Fellow, Dr. Weir Mitchell, who first called special attention to it, and gave it a name, in 1874;* but already in 1873,† Professor Charcot, of Paris, had quite well described the movements, and indicated their association with hemi-anæsthesia. I would here beg to call attention to the fact that the description of the symptoms in my first case was written in December, 1873. Recently M. Charcot has written specially upon "post-hemiplegic" chorea in its relations to hemi-anæsthesia,‡ and has expressed the opinion that both these symptoms are caused by lesions situated at the outer and posterior borders of the optic thalamus, or in the posterior expansion of the corona radiata. Last year,§ Dr. W. R. Gowers read to the Royal Medical

* Post-Paralytic Chorea. American Journal of the Med. Sci., Oct., 1874, p. 342.

† Leçons sur les maladies du systême nerveux, t. i., p. 279.

‡ Leçons, etc., 4me fascicule, Paris, 1877, p. 329.

§ On Athetosis and Post-hemiplegic Disorders of Movements. The Lancet, 1876, vol. i., p. 709.

and Chirurgical Society an elaborate essay based upon a number of cases showing various degrees of post-paralytic incoordination, including athetosis, athetoid movements, and post-hemiplegic chorea.

A good description of a remarkable case of this condition, also coincident with hemi-anæsthesia, will be found in M. Schœpfer's Paris thesis.*

I now pass on to relate the cases which have come under my observation.

CASE I.—H. H. H., aged eighteen years; by occupation a clerk, and a native of this country, was referred to me by Dr. Fisher, December 6, 1873.

He was a fairly-developed boy, with apparent good general health. He had been well until last April, when he began to suffer from diffused headache, more temporal, but not one-sided. One day in May he went down to his office feeling as well as usual, and began his work with full use of all his limbs. About 11 o'clock A.M. was writing, leaning on the desk, the left arm thrown forward and its fingers steadying the paper. He got up to cross the room with a book in his left hand, but the book fell to the floor, and he then first became aware that his left arm was weak. There were at the time no subjective or sensory symptoms, cerebral or peripheral. The leg was not in any way affected. He was able, though awkwardly, to feed himself at dinner that evening.

The paresis gradually increased until the middle of July, and since that time there has not been much change.

Shortly after the attack, within a few days, patient noticed numbness of the left hand and forearm, and this has somewhat increased.

In the early part of July convulsive movements began in the left hand, and have since become greater in force, and have extended to other parts of the left side. The left leg began to twitch and grow weak also in the first part of July, and since October slight twitching has appeared in the lower part of the left side of the face. Lately sight has become impaired, but hearing is preserved.

There have never been any symptoms on the right side. He has never had any epileptic or epileptiform seizures. Memory has somewhat failed.

* Considérations sur un cas d'hémianesthésie avec mouvements ataxiques, succédant à une hémiplégie du même côté. Thèse de Paris, 1876.

In the middle of July there appeared double vision, which gradually passed away. At the same time he had severe bi-temporal headache with nausea, lasting one week. The latter symptom never recurred, and it was probably caused by diplopia. No rectal or vesical symptoms. No dysphagia.

EXAMINATION reveals left hemiplegia with peculiar spasmodic movements of the palsied parts. The left arm and leg execute all movements, though feebly and awkwardly. The dynamometer shows a strength of 30° in the right hand and 10° in the left; can barely stand on left foot alone; no evident facial paralysis; tongue points a little to the left. There is now no twitching in the face. The left pupil is a trifle larger than the right; both are active. The ophthalmoscope shows both optic disks congested, their outlines blurred, and traces of exudation along the bloodvessels. There is no evident palsy of any muscle about the eyes, though the eyes converge abnormally. Fields of vision not impaired.

There are strong *choreiform* (this is the word used in notes made at the time of first seeing patient—1873) movements in the left arm, shoulder, and leg. These parts are constantly agitated during waking hours, but quiet during sleep. The arm is more or less rigid, and its various parts perform large oscillations. The movement is increased by emotion or by the attempt to perform voluntary acts; he cannot carry a glass to his lips. Eyes being closed, he can, after groping a little, place fore-finger on tip of nose. There is, however, a marked ataxiform element in the spasm. The muscles of the shoulder and the trapezius are involved, but not the muscles of the trunk and neck.

Sensibility (to contact and pain) is slightly impaired in fingers and hands; the points of the æsthesiometer must be separated from 3 to 5 mm. to be distinguished on the finger-tips.

January 20th, 1874.—Chorea is as before. There is much hebetude. On the right side there is marked ptosis with weakness of the internal rectus (palsy of third nerve).

We now have a form of *crossed* paralysis, *i. e.*, third nerve on the right side, body on the left. The right crus cerebri is probably involved in lesion.

January 28th.—Greater evidence of palsy of third nerve on right side; more chorea in left limbs; left side of face paralyzed; speech thick, saliva escaping from the mouth. There is marked anæsthesia in range of left supra-orbital nerve. Sister states that he

has frequent partial syncopal attacks without spasm or loss of consciousness. Is this petit-mal ?

March 1st.—Much as before, but weaker. While speaking has spasm in left side of face which simulates involuntary laughter.

This patient died during March without presenting any new symptoms, and I was unable to make an autopsy.

Judging by the symptoms, the lesion must have been placed just above the motor tract of the right crus cerebri, acting upon it by pressure.

It is probable that the lesion was hemorrhage with subsequent inflammation about the clot, though it may have been a tumor. I am in doubt on this point, because I am not disposed to attach much value to the ophthalmoscopic examination which I made. The certain diagnosis of neuro-retinitis would, of course, have strengthened the probability of there being a tumor in the locality indicated.

CASE II.—J. P., aged 26, a clerk, was referred to me by Prof. Edward Curtis, on May 24th, 1877.

I learned that this young man had been well up to April 16, 1876, when he had, in the street, an attack of right hemiplegia. He did not fall down ; was able to walk with help to a street-car, yet knew nothing of what afterward occurred until the next day. This was either late loss of consciousness, or amnesia.

On the next day speech was much affected, he could not recall names and addresses of persons ; his right arm was powerless, and there was much numbness of the right cheek and arm. "Could only see with inner half of right eye." Right leg weak. In a day or two speech improved.

In three weeks he walked well, but the arm did not regain its strength for three months.

During this period of improvement irregular movements appeared in the right arm, and have persisted. No jerking in face, leg, or on the left side of the body. Imperfect vision persisted. The numbness continued marked in toes, finger-tips, cheek, and tongue, on the right side. The numbness on the right side of face and tongue never quite reached the median line.

In August (four months after attack of paralysis) had an epileptiform spasm, followed by loss of consciousness lasting until the ensuing morning.

In April, 1877, had a second epileptiform attack. At times

feels queerly in his head, "as if a spasm were coming on." Has not suffered from headache or dizziness.

Inquiries into patient's past history show that six or seven years ago he had several chancres, which did not heal for three months. Had no buboes. Never had any eruptions, strictly speaking; but his legs "ulcerated," and he has since frequently suffered from ulcerated sore throat. At no time any osteocopic pains. Sight was good until after attack of paralysis. Memory is a little impaired.

EXAMINATION.—Patient's speech is imperfect, a sort of aphasic stammering. It would appear that he has always had a somewhat similar defect. The right leg has almost perfectly recovered, but the right hand is not quite as strong as the left, and presents curious abnormalities of movements. The dynamometer test shows for the right hand 18°–22°; for the left, 28° and 28°.

The peculiar movements are of two sorts:

1. While the patient is seated quietly, with his hand resting on his thigh, it (the hand) may be seen to be agitated by slight rythmical movements of the type observed in paralysis agitans. These are not truly constant, but seem to be provoked by observation or by the patient's own watching of the hand.

2. During attempted use of the extremity, an ataxiform movement is developed; or, more properly speaking, a movement compounded of the want of combined and harmonious action of large muscular groups which is characteristic of the ataxic movement, and of the totally irregular and capricious muscular contractions which constitute the choreic type. On the whole, the movement is more like that of ataxia; *i. e.*, more or less regularly oscillatory.

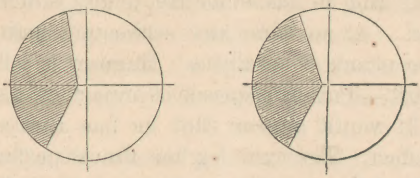
The right leg is not the seat of any tremor, ataxia, or chorea; and the left side of the body shows none.

There is no marked facial palsy, but the right naso-labial crease is less distinct than the left. The tongue deviates a little to the right. These points make it probable that the paralytic attack in April, 1876, was one of common typical hemiplegia.

There exists a degree of right hemi-anæsthesia. To slightest contact, according to patient's positive statement, the anæsthetic district of the face begins at a point 1 cent. to the right of the median line. The æsthesiometer and the simple contact test show slight loss of sensibility on the right side of the face. On the finger-tips the points of the æsthesiometer cannot be distinguished at a less distance apart than from 5 to 8 mm. on the

right side, while on the left they are distinguished at 3 mm. Pricking is well felt.

The fault in vision is in the shape of incomplete hemiopia; the right temporal and the left nasal halves of the fields being obscured, as shown in the cut.



RIGHT EYE. LEFT EYE.

The pupils are equal and normal; the ocular muscles act well; and the ophthalmoscope shows no lesion which I can appreciate.

A careful examination of the throat does not reveal any cicatrices. The heart is normal.

My diagnosis was slight cerebral hemorrhage just outside the left thalamus opticus. Yet I was unwilling not to give the patient the benefit of the doubt that syphilis had led to arterial disease and rupture, though the history was not one that pointed to the existence of syphilis at any time. Yet how often are serious syphilitic nervous lesions developed after equally or more incomplete chains of evidence? Consequently I have given this patient mercury internally, and have brought about a degree of salivation.*

REMARKS.—These cases are strongly in support of M. Charcot's proposition. In the first place, we observe in both the co-existence of hemiplegia, hemi-anæsthesia, and choreiform movements. In case I., a positive symptom, palsy of oculo-motorius nerve, points to a lesion near the crus cerebri—probably just above it. In the second, there is a strong probability that the lesion is not far from the same part—near the thalamus opticus.

In my second case, the occurrence of hemiopia is of great interest, because it will be remembered that Prof. Charcot has recently† denied that a lesion of the hemisphere could produce

* *July* 1, 1877.—I may now add that salivation, followed by the administration of iodide of potassium in doses of 3 iii. a day, made no change whatever in this patient's symptoms. I have now advised him to cease treatment, and hope for spontaneous improvement.

† Leçons sur les localisations dans les maladies du cerveau. Paris, 1876, p. 126.

hemiopia. In the absence of a post-mortem examination, I would not be understood as claiming that the case positively contradicts M. Charcot's statement; yet it must be admitted, I think, that there is no probability that there was in this case a second lesion affecting one of the optic tracts.

CONTRIBUTION

TO THE

STUDY OF LOCALIZED CEREBRAL LESIONS.

By E. C. SEGUIN, M.D., of New York.

It has fallen to my lot to observe during life and to examine after death a number of cases in which localized cerebral lesions gave rise to definite peripheral symptoms, and it has appeared to me that these cases might profitably be studied in the light of recent experimental and pathological researches upon the functions of the brain. In other words, I shall endeavor to determine the bearing of these cases upon the recent hypothesis of the localization of functions in the cortex of the brain.

I shall divide my cases into three categories: 1st. Cases in which localized lesions gave rise to Aphasia. 2d. Cases in which localized lesions gave rise to Paralysis. 3d. Cases in which localized lesions gave rise to Spasm.

PART I.

CASES IN WHICH A MORE OR LESS LIMITED CEREBRAL LESION PRODUCED APHASIA.

Case I.—*Cerebral softening from arterial degeneration: aphasia, and right hemiplegia.*

A woman aged seventy-five years was admitted to the Epileptic and Paralytic Hospital on Blackwell's Island, January 17th, 1873. The history of the case is very meagre, and only states that when attacked she screamed, threw up her hands, and became insensible. On recovering from insensibility it was found that she had com-

pletely lost speech, and was paralyzed in the right side, face, and limbs. No mention is made of the state of sensibility; but, as I am in the habit of always looking for hemi-anæsthesia, I feel sure that the case was one of common hemiplegia. I also feel very positive that during the six months of the patient's stay in my ward she greatly regained voluntary power on the right side, being able to take a few steps alone, and to move her arm quite freely. Aphasia, however, remained complete.

The patient died on July 12th, 1873, and careful notes were made at the time of the post-mortem examination on the 13th. I reproduce only those relating to the state of the brain, making, however, the preliminary statement that the valves of the heart were free from disease.

Dura mater not abnormally adherent to the skull, and healthy at base. Moderate sub-arachnoid effusion at the top of the brain, and a great deal at its base.

Both anterior lobes have undergone more or less atrophy. The convolutions of the left parietal lobe, beginning at a point an inch and a half from the median line, bulge and form a soft and yellowish tumor. The pale yellowish color of the affected convolutions is in marked contrast with the injected appearance of the rest of the brain. Over these diseased convolutions there is more sub-arachnoid effusion than elsewhere. After removal of the brain, and escape of the greater part of the serum, the left parietal lobe appears sunken, and there results an appearance like a loss of substance as large as a small walnut. The basilar and other large arteries of the circle of Willis are open, and nearly free from changes.

Careful inspection shows no externally visible lesion in the left frontal lobe, its third convolution, and the island of Reil. The depression in the parietal lobe measures six and a half cent. square, involving all of the lobe except the part lying next to the great longitudinal fissure.

Two small old lesions are found in the right hemisphere, one lying at the external margin of the extra-ventricular nucleus of the corpus striatum, just behind the island of Reil; the other in the posterior part of the same (lenticular) nucleus. Both are small cavities, the size of large beans.

The following is a transcript of notes which I made upon the state of the left hemisphere. I was very careful in studying the limits of the *ramollissement*, because at first sight the case seemed

to be one opposed to Broca's hypothesis of aphasia. The extent of the lesion was studied, after hardening for a few days in a solution of bichromate of potassa, by means of transverse horizontal sections, and tracings made on transfer paper upon each section by Mr. George Wright. Each tracing was afterward carefully reduced and drawn to a scale, with a sketch of the appearance of the unsliced hemisphere. This last drawing, Fig. 1, shows that the greatest destruction of tissue has taken place in the parietal lobe proper. Posteriorly and inferiorly a degree of degeneration of

CASE I.—PROFILE OF LEFT HEMISPHERE. TWO TRANSVERSE HORIZONTAL SECTIONS OF SAME. EXTENSIVE RAMOLLISSEMENT.

the convolutions may be traced almost to the confines of the occipital and sphenoidal lobes; while anteriorly, after having destroyed in great part the ascending frontal convolution, it extends over a portion of the second frontal gyrus.

Sections were made through the hemisphere in the planes indi-

cated by lines in Fig. 1, and the morbid appearances seen on each section were accurately traced on paper. Fig. 2 shows that the atrophy has destroyed a large part of the upper posterior part of the hemisphere. Fig. 3 demonstrates that the hinder part of the third frontal convolution is likewise involved.

By this method of examination the case is restored to the category of common cases of aphasia, viz., those supporting Broca's hypothesis.

Remarks.—I consider this case as doubly instructive.

First, in a positive manner, as showing that a superficial degeneration of the cortex of the brain (involving Broca's centre for speech) may produce aphasia of the most complete kind.

Second, negatively; because, in spite of the destruction of gray matter in the regions which Ferrier makes out to include nearly all the motor centres for the face, arm, and leg, a great degree of voluntary power was regained by the patient in a few months. Hence it would appear that other parts of the injured hemisphere had acquired controlling power over the limbs of the opposite side.

Case II.—*Embolism of the left middle cerebral artery, softening of the brain: aphasia, and right hemiplegia.*

Mrs. G., seen in consultation with Dr. William Pierson, jr., of Orange, N. J., on May 8th, 1877. With the exception of a badly acting heart and a tendency to gout, Mrs. G. enjoyed fair health and had three children up to 1875. Late in November of that year she had a moderately severe miscarriage. On December 7th, at 7.30 P. M., experienced a sudden attack of common hemiplegia on the left side, without loss of consciousness. Speech was much impaired by defect in articulation. The palsy passed away rapidly, though she dragged her left foot for some days. Power of articulation slowly regained. Patient's husband is an unusually well-informed gentleman, and states that trouble in speech was surely not aphasiform. There remained a want of proper action of muscles of throat and larynx; patient not knowing how to pitch her voice afterward.

On January 13th, 1876, a second attack of left hemiplegia occurred. Consciousness was lost for a moment. Left leg but not left arm palsied. All symptoms, except a degree of excitement, passed off the next day, after a long sleep.

On April 1st, at 1 A. M., had a first epileptic fit while asleep; had full spasm, slight frothing at mouth, loss of conscious-

ness, stertorous breathing, and a heavy terminal slumber. On April 29th, second fit; falling like a shot to the floor. Has had a few attacks since; only two, however, since last August. One was two weeks ago.

Present paralytic attack occurred twelve days ago. On April 26th, came down stairs, and in a few minutes was found fumbling a door-knob. Without apoplectic symptoms, complete right hemiplegia and aphasia were developed. Possibly a little return of voluntary movement has taken place in the right limbs. No return of speech. Indeed, the aphasia has been so absolute, that even sign-language has been lost, and patient's friends have thought her deprived of her faculties, though she has been conscious at all times. There has been no retention of urine, no tendency to bed-sore. Food has hardly been taken, and patient's respirations have been extraordinarily frequent.

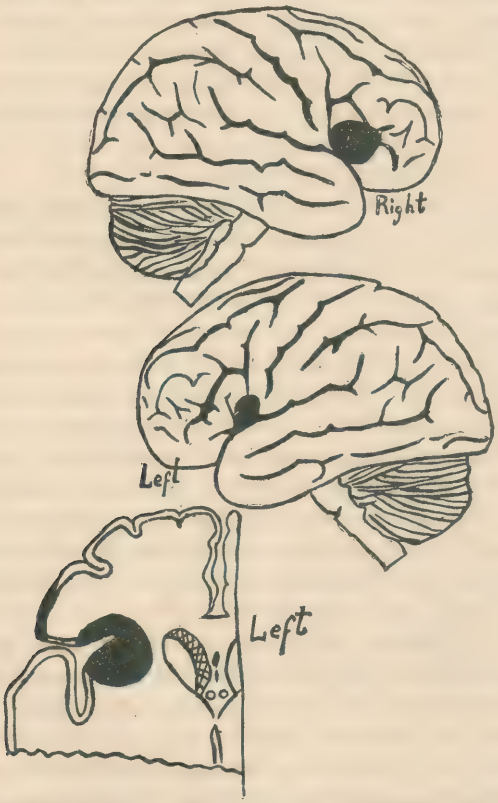
Examination shows complete right hemiplegia, with marked loss of sensibility in the paralyzed limbs. The face is hardly distorted. Absence of spoken and gesture language as above, but patient looks intelligent. Pupils very wide, but equal. Breathing very rapid, 50 or 60 in the minute. Pulse slightly irregular, beating about 90 in the minute. The heart is enlarged, apex in sixth intercostal; cardiac sound can be heard away from the patient, as far as two feet. No distinct murmur can be heard, but the heart-sounds are completely reduplicated. Mouth temperature 37.5° C. No sensibility to pinching on the right side, and only slight on left; but this may be owing to rapid respiration. There is a marked tendency to turn eyeballs to the left, away from the paralyzed side. Lungs are free from deposit or congestion; the optic disks are normal.

I learn that Mrs. G. has had pains in the small joints, with atrophy of hand-muscles, and, in the last few years, gouty knees. From childhood she has had a curious affection of the heart, characterized by dyspnœa, hard beating of the organ, and by transmission of its sound to a distance from the body of two to four feet. My diagnosis was embolism of various arteries in both hemispheres; the last attack being due to blocking of a branch, or branches, of the left Sylvian artery, and consequent softening of (probably) the third frontal and the ascending frontal convolutions.

In spite of an attempt at supporting treatment, Mrs. G. died on May 11th.

For notes of the post-mortem examination, made on the same day, six hours after death, I am indebted to Dr. Pierson.

"The membranes and sinuses of the brain were normal. The surface of the third frontal convolution of the right side was yellow in color, with a tough, elastic feel. This lesion, extending through the gray matter, was about one inch square and one-quarter inch thick. The same change was also found, but to a less extent, in the same part on the left side. The left middle cerebral artery was plugged by an embolus at a point one-half



CASE II.—SOFTENING IN BOTH HEMISPHERES, IN CONSEQUENCE OF EMBOLISM.

inch from its origin. A spot of brain-tissue, about the size of a quarter of a dollar (one inch in diameter), supplied by this artery, was in a softened state; it was yellow, tinged with red, broken down, and of semi-fluid consistence. The situation of this lesion was external and posterior to the anterior cornu of the

left lateral ventricle, and corresponded to that part of the third frontal convolution which is known as the island of Reil. The softening was confined to the white brain substance, and did not implicate the surface. The rest of the encephalon was normal.

"The heart was much diseased. The mitral valves were much thickened, and nodules of atheroma could be felt at various points. The two leaves of the valve were joined together, constricting the auriculo-ventricular opening to such an extent that it would not admit the end of the finger; there was stenosis and insufficiency."

Unfortunately the brain was not preserved for more minute examination, so that the existence of microscopic changes in the rest of the territory supplied by the left Sylvian artery remains a matter for speculation.

The notes and the plugged artery were brought to me by a student of Dr. Pierson, and I asked him to mark out for me on plates in Ecker's and Luys' books the location of lesions. The accompanying diagrams are constructed faithfully after this gentleman's indications, and Dr. Pierson's corrections.

Remarks.—The complete aphasia in this case was undoubtedly due to lesion of the third convolution and of the anterior folds of the island of Reil. We have in this case an addition to the large list of cases supporting Broca's hypothesis. It is remarkable that such complete hemiplegia should have existed without apparent lesion of those parts which are more directly connected with the movements of the arm and leg, viz., the ascending frontal and parietal convolutions. These convolutions are supplied by the artery which was plugged, and it is very probable that microscopic study would have shown them to be full of granular bodies. They may have been saved from gross softening by an unusually free anastomosis between the final branches of the plugged Sylvian artery and other arteries in the same hemisphere. It is also possible that a part of the hemiplegia was due to pressure upon and anæmia of parts adjacent to the softened spot; and that, had the patient survived, the paralysis would have disappeared in a few weeks.

Another interesting feature of the case lies in the connection between the very localized lesion in the right hemisphere and the first attack of hemiplegia. This attack, it will be remembered, was characterized by slight and transitory paralysis of the limbs, and by very great defect in articulation. Indeed, it is said that

the patient never recovered the full use of her vocal organs. From this we might be led to infer that the function of the third frontal convolution on the right side in right-handed human beings is intimately connected with the muscles governing the movements of articulation and phonation.

CASE III.—*Chronic meningitis almost limited to the posterior part of the third left frontal convolution, extensive central cerebral softening: epilepsy, chronic aphasia of varying degree, right hemiplegia; death in status epilepticus.*

A male, age forty-seven years, was admitted to the Connecticut General Hospital for the Insane, at Middletown, on Nov. 29th, 1873.

For a full account of the case and for the post-mortem examination I am indebted to my friends Drs. A. M. Shew and W. B. Hallock, medical officers of the hospital.

The patient was sent to the hospital because of "dementia." It was learned from his wife that he had had three "apoplectic" attacks. The first occurred in April, 1873, and consisted in loss of consciousness and general spasm. After this he had at short intervals some few slight "spasms." In July had a second severe attack of same kind as the first, followed by slight hemiplegia. Three weeks later had a third seizure, after which he was in a state of delirious mania, which still continues.

On admission, mind is in dementia, the pupils are very small, articulation good, the tongue protrudes straight, and the only palsy apparent is in right lower limb. At times is violent and has insomnia. Speech very incoherent. In December it is noted that palsy is less, but that legs are not well co-ordinated, and that hands tremble.

In middle of January, 1874, patient is calmer, and more rational, but speech is imperfect. "He talks plainer, seems to have ideas, but has forgotten words." Aphasia.

Feb. 17th.—At two o'clock P.M. had an epileptic attack, with paralysis of right side; tongue deviating to the right. Four other spasms in three-quarters of an hour; each characterized by usual symptoms, foaming at mouth, stertor, upturned eyeballs.

March 6th.—Is up and about the ward. Is more coherent than for some time. Hallucinations of sight in the night.

March 30th.—Is discharged improved. For some time past the difficulty in speech has taken the form of amnesia, and his writing has shown the same characteristics.

Re-admitted June 29th, 1874. Aphasia still present, and he

seems to appreciate the trouble. "I like that fountain I drink from three times a week," meaning, I like that medicine I take three times a day. He knows that he calls things by wrong names, and is thereby irritated. "I wish you would sell me," meaning bleed me. "Oh, I can't talk."

July 26th.—Yesterday and last night had ten attacks of an apoplectiform character, without paralysis. These must have been epileptic seizures.

Sept. 29th.—Rose as usual, made up his bed nicely. At table was unable to grasp knife and fork. In twenty minutes, complained of bad smell and dim vision, then had a general convulsion. Had ten attacks in the course of an hour. There was foam at the mouth, and more twitching on the right side of the body. In a few days was up again.

Oct. 20th.—Three epileptic attacks in a few minutes. This is the third time that patient has had prodromata, consisting of nervousness, a slight degree of paralysis of right side; has gone to his room and made signs that he was ill.

Oct. 21st.—No paralysis after attacks; some excitement in night.

Nov. 19th.—Is in good physical health; has hallucinations of sight; aphasia continues marked.

Jan. 8th, 1875.—After dinner (1 P.M.) had an epileptic seizure, and at 5 P.M. the attacks followed each other rapidly. The right side is completely paralyzed.

Jan. 9th.—Has been in status epilepticus all night, and must have had nearly four hundred convulsions. Died at 3.15 A.M.

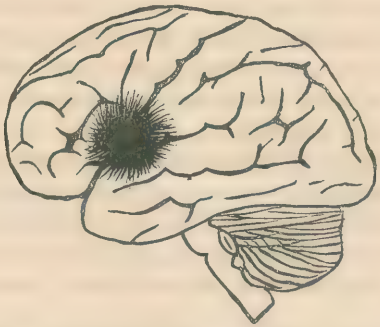
Autopsy Jan. 10th, thirty-two hours post mortem. Calvarium normal. Vessels of dura mater and of pia mater congested. Dura mater adherent to bone over right posterior lobe of cerebrum, membranes adherent to pia mater over left middle lobe of brain. General moderate opalescence of the arachnoid. Other organs in remarkably good condition.

The entire brain was at once sent to me in a solution of bichromate of potassa, and I made a careful examination of it in a few days.

The only externally visible lesion is on the left hemisphere. There a patch of dura mater adheres to the soft membranes and the brain, at the posterior extremity of the third frontal convolution and the lower extremities of the ascending frontal and ascending parietal convolutions, bridging over the fissure of

Sylvius. The patch measures 22 mm. in a vertical direction, and 15 mm. horizontally. The anterior border of the piece of membrane is 58 mm. from the apex of the frontal lobe; its upper edge is 58 mm. below the upper border of the frontal lobe (at the longitudinal fissure); and its lower border, lying on the fissure of Sylvius, is 55 mm. above the lowest part of the sphenoidal lobe.

The arachnoid and pia mater surrounding the patch, in the fissure of Sylvius and for 25 mm. above it, are thickened and whitish.



CASE III.—PATCH OF PACHYMENINGITIS ADHERENT TO LEFT HEMISPHERE.

The patch lies in a depression in the underlying convolutions, and a horizontal transverse section through it and the hemisphere shows that the three membranes are fused in the patch, which is 2.5 mm. thick, and that the subjacent gray matter of the third and ascending frontal convolutions, and the ascending parietal convolutions, are grayish and translucent.

The same section reveals extensive softening in the central parts of the left hemisphere. The lenticular ganglion (extraventricular part of corpus striatum) is soft and reddish; contains nervous *débris*, granular bodies, and granular blood-vessels. The few ganglion cells seen are filled with granulations.

A second softened spot is in the white centre of the hemisphere, in its posterior half, outside and above the lateral ventricles. The microscope shows the same granular detritus as in lenticular ganglion. Abundant granular bodies are also found in the white centre of the frontal lobe, and in the convolutions of the island of Reil, deep in the fissure of Sylvius.

The corpus striatum proper contains only a few granular ganglion cells.

Microscopic examination gave evidence of descending degener-

ation in the motor tract through the left half of the pons Varolii and medulla oblongata, and the right lateral column of the spinal cord.

The ganglion cells of the anterior horns of the spinal cord contained an abnormal amount of granular matter, and the posterior columns contained an immense number of amyloid bodies.

REMARKS.—This case, partly from its complicated nature, and partly because of its imperfect record, is difficult to analyze with reference to the question which I have in view in this contribution.

In addition to epileptic, paralytic, and aphasic manifestations, there seems to have been actual insanity present, as manifested during life by hallucinations and delusions, and by incoherence and dementia; and post mortem, by opacity of the delicate membranes of the brain, and degeneration of the posterior columns of the spinal cord.

It seems probable, however, that some of the so-called "dementia" in the first part of the history of the case was aphasia.

In estimating the share of the two lesions in the production of symptoms, it must be admitted that, whereas the patch must have been ancient, the *ramollissement* cannot have been very old. The latter lesion was, however, at least six weeks old, since secondary descending degeneration had set in to a slight degree. Yet, as the chief phenomena of the disease—epileptic seizures, transitory hemiplegia, and aphasia—existed from the beginning of the illness, it is right to conclude that the older lesion, *i. e.*, the localized chronic meningitis, was the cause of these symptoms.

The thickened state of the pia mater, arachnoid, and dura mater, acted upon subjacent parts in several ways: by pressure and by mechanical irritation, increased by the respiratory and cardiac movements of the brain, and by interfering with the blood-supply of the gray matter, and of the white substance for a certain depth. The part which was most affected by this lesion was Broca's speech centre in the posterior part of the third frontal convolution.

Consequently, it seems right to me to consider this case as favorable to the hypothesis of the localization of functions in limited parts of the cortex of the brain.

As to the nature of the localized meningitis, nothing is said in the history of the case concerning syphilis, or symptoms belonging to the syphilitic category. Sections of the patch and subjacent nervous tissue were very difficult to make, because of the

difference of density between the two tissues and the lax bond o. union between them. The specimens now passed around must be judged leniently because of these peculiarities. In so far as the thickness of the sections will permit microscopic study, it seems to me that the lesion is a simple hyperplastic one, having resulted in the formation of dense fibrillar connective tissue. In no part of the specimens can I find the numerous young cells so characteristic of gummatous products.

CASE IV.—*Constitutional syphilis, commencing caries of the dorsal vertebræ, acute tuberculosis, tubercular meningitis, most developed over left third frontal convolution and island of Reil: intermittent aphasia, and later, hemiplegia.*

Mr. X——, aged forty-three years; a private patient of Drs. William H. Draper and Frank P. Kinnicutt. The latter gentleman has kindly furnished me with abbreviated notes of the cases, and the brain was placed in my hands for examination.

The patient was a victim of unusually severe syphilitic infection: having had series of secondary and tertiary lesions while under Dr. Draper's care. At the time of his last illness he had syphilitic neuralgia. In 1874–5, there was slight trouble at apex of right lung, but the disease seemed wholly arrested during the past year.

Since two months, emaciation has rapidly advanced, and strength has much diminished. Since April 20th, there has been fever of a very irregular type; the temperature varying from 37.1° to 39.1° C.; the temperature not being the same on any two days. Pulse has been very frequent: 100 to 120. Physical examination has revealed simply moderately fine moist *râles*, at first only in the anterior and posterior parts of the right lung; later, during the last four weeks of life, in both lungs.

On May 10th, there was suddenly developed aphasia and agraphia, without loss of consciousness or paralysis. [This negative statement is not made upon the patient's or the nurse's statement, but after critical examination by Dr. Kinnicutt, whose accuracy in clinical observation is extreme.] This condition of aphasia continued about twenty-four hours, and during this time the patient's mind was perfectly clear. A nearly complete intermission (return of speech) then occurred, followed in twenty-four hours more by a second attack of complete aphasia and agraphia, also without impairment of consciousness or mental clearness, and without paralysis. These intermissions and attacks con-

tinued to succeed one another until within forty-eight hours of death, when the intermissions became incomplete. At first, there existed only a certain slowness of speech during the intermissions; but in the last few days there was partial aphasia; or, more properly speaking, aphasia would show itself after a few moments of correct speaking. Right hemiplegia was almost imperceptibly developed (face and limbs); became marked forty-eight hours before death, and was complete, with a semi-comatose state at the last.

At no time was there only pain in the head. General hyperalgesia (more marked on the right half of the body) was present in last week of life.

Dr. Kinnicutt diagnosticated acute pulmonary tuberculosis, with a cerebral complication. There was a doubt in our minds, whether the cerebral lesion was meningitis, tubercular or syphilitic, or whether it consisted in syphilitic arteritis, involving the left middle cerebral artery.

The post-mortem examination was made on May ——, sixteen hours after death, by Dr. Kinnicutt, and the following lesions found:

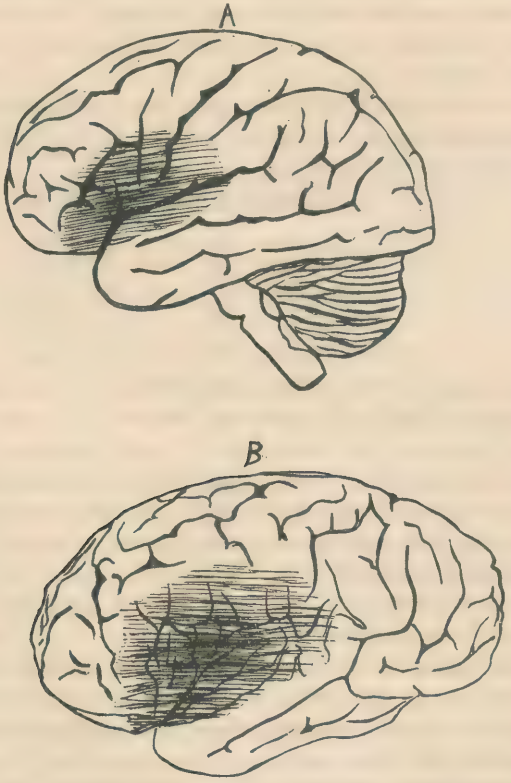
There was a cavity about three-quarters of an inch in diameter, filled with pus, in the right half of the body of the twelfth dorsal vertebra; and the pus was seen to have made its way within the sheath of the psoas muscle, nearly as far as Poupart's ligament. At the apex of the right lung was a dense cicatrix, and over one of the lower ribs on the left side, the remains of a gumma, which had suppurated many months. Gray tubercular granulations were found in abundance throughout both lungs, in the spleen and kidneys. There were none in the liver, and their presence in the peritoneum was doubtful.

The brain, placed in a solution of bichromate of potassa, was at once sent down to me for examination. I did not do more at first than note that the left sphenoidal lobe had been damaged in the removal of the brain, and that there were a very few granulations, the size of tobacco seeds, or a little larger, in the pia mater on the convexity of the hemispheres. My reason for not cutting the brain while fresh was that, from the lack of consistency of the left anterior lobe, Dr. Kinnicutt thought it likely that there was an abscess or a patch of *ramollissement* near the third frontal convolution, and I wished, if there were such a lesion, to obtain its exact topography.

The brain having been completely hardened in bichromate of

potassa, I proceeded to examine it by means of vertical and horizontal sections. Somewhat to my surprise, I found no lesion in the deeper parts of the hemispheres. There was only a lepto-meningitis of very peculiar distribution.

In the first place, the convexity and base, and the whole of the right hemisphere, showed only traces of exudation alongside of the chief vessels of the pia mater, with here and there a granulation varying in size from .5 mm. to 1 mm. in diameter.



CASE IV.—A, SHOWING FOCUS OF TUBERCULAR MENINGITIS ON EXTERNAL PART OF LEFT HEMISPHERE. B, THE EXUDATION IN THE FISSURE OF SYLVIUS.

In the second place, the lower median region of the left hemisphere showed very much more developed exudation. The vessels covering the posterior part of the third convolution as it dips into the fissure of Sylvius were bordered by thick bands of exudation quite as wide as the vessel itself, and the pia mater was thickened over a space 25 mm. or more in diameter. On opening the

fissure of Sylvius, the pia lying in it was found enormously thickened, and its meshes filled with semi-solid and solid exudation.

The focus of the meningitis was in the territory of distribution of the left middle cerebral artery, especially over the third frontal convolution, and the convolutions of the island of Reil.

Microscopic examination of the exudation showed an accumulation of young cells in the meshes of the pia mater, and more especially round about blood-vessels next the cortex. Around many of these, the exudation formed tumor-like swellings, or, more exactly speaking, muff-like masses. The young cells could be followed some distance into the cortex of the brain, lying in the perivascular spaces. This examination corroborates the diagnosis of tubercular meningitis.

PART II.

CASES IN WHICH A LIMITED CEREBRAL LESION CAUSED PARALYSIS.

CASE V.—*Limited softening of the left ascending frontal convolution; right hemiplegia without aphasia.*

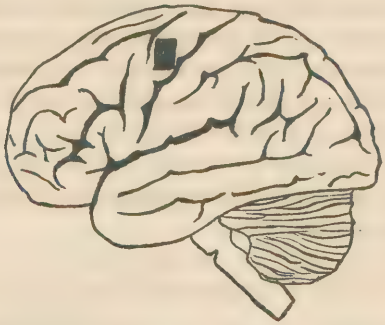
A female, aged fifty-four years, was admitted to my ward in the Epileptic and Paralytic Hospital on Blackwell's Island, on June 20th, 1875.

The brief history of her case is to the effect, that about Christmas, 1874, during the night, she was suddenly paralyzed on the right side. She did not lose consciousness, her face was not paralyzed, and speech was preserved.

Examination shows that patient's face is not paralyzed, her tongue points straight, the right upper extremity is palsied, but there is slight voluntary motion at elbow and fingers. The lower limbs are without voluntary motion. There is some œdema about the ankles, and a superficial bed-sore on both nates. Death occurred June 23d.

Post-mortem examination. The only externally visible lesion in the brain is a disappearance of a part of the convolution in front of the fissure of Rolando, within 25 m.m. of the great longitudinal fissure, on the left hemisphere. It is not like a yellow patch, but is more like an ulceration of the convolution. A horizontal section, made through the hemispheres above the ventricles, shows healthy tissue in the right hemisphere, while in the left it reveals a softened but not much discolored part in con-

nection with the superficial lesion above described. A vertical transverse section through this lesion and the lower part of the left hemisphere shows that the softened mass has its greatest diameter vertically, and extends from the roof of the lateral ventricle upward.



CASE V.—LIMITED SOFTENING OF THE LEFT ASCENDING FRONTAL CONVOLUTION.

No other lesion is found in the cerebral tissue. The cerebral arteries show some arteritis, especially the left middle cerebral; but their channels are open.

A considerable spinal lesion is found, consisting in purulent infiltration of the lower cervical muscles alongside the 3d, 4th, and 5th vertebræ. On removing the posterior portion of the vertebræ, there is found a thick exudation, like a membrane, between the dura mater and the bones. Between the dura mater and the bodies of the vertebræ there is no trace of exudation. The arachnoid and the spinal cord seem healthy. The only osseous lesion is a roughness of the arches of the cervical vertebræ near the exudation.

This case is only to be made use of with reservation, because of its imperfect history, and because of the co-existence of a spinal peri-pachymeningitis. The main interest of the case is a negative one of unquestionable exactness. At no time was there aphasia; and it may be seen that the softened patch was placed quite far from the speech centre, above and behind it. It would seem that the face and tongue were not palsied; and this again is in accord with the location of the lesion, which is in the ascending frontal convolution, near Ferrier's centre, No. 6, which he* finds in the monkey, to co-ordinate certain movements of the arm and forearm.

* The Functions of the Brain. New York, 1876; p. 306.

PART III.

CASES IN WHICH LOCALIZED CEREBRAL LESIONS GAVE RISE TO LOCALIZED CONVULSIONS OR SPASM.

CASE VI.—*Injury to the top of the skull on the right side, osteitis, inflammation of the dura mater, with lesion of subjacent convolutions; development of a large sarcomatous tumor in the right hemisphere; epilepsy; spasms limited to left arm, neck, and face; left hemiplegia: no neuro-retinitis.*

George S., a laborer, was admitted to my service in the Epileptic and Paralytic Hospital, Blackwell's Island, on April 19, 1875, for epilepsy.

The following history of his case was obtained:

On December 19, 1869, he got out of bed, in the middle of the night, to get some water; went to a stairway on the outside of the house, made a misstep, and fell to the ground. He lay insensible until morning, when he was taken care of. Paralysis of the left side followed the injury, but improved under treatment sufficiently to allow him to do his ordinary work.

Three years later (1872), he was seized with epileptic convulsions of the common typical sort: sudden fall, general spasm, biting of tongue. These attacks ceased in December, 1874, and were then wholly replaced by very frequent attacks of partial or localized epilepsy, without loss of consciousness.

These attacks consist of tonico-clonic spasm of the muscles of the left side of the face and neck, and of left upper extremity, especially the thumb and index. The left upper extremity is strongly flexed and the mouth drawn to the left during the attack, which lasts from 80 to 140 seconds without nitrite of amyl, and from 70 to 90 seconds with it. The attacks occur with extreme frequency, from three to eleven taking place every hour. At night they are rare. The spasm begins simultaneously in the facial muscles and in those governing the thumb and index. Consciousness is never lost. No general convulsions occur. An examination shows the left pupil to be a trifle larger than the right; the left cheek is paretic; the left arm and forearm absolutely paralyzed, and the left leg weak. The gait is characteristic of hemiplegia. There is marked tactile anæsthesia on the left side; the two points of an æsthesiometer not being distinguished at 45 mm. on the forehead, at 40 mm. under the eye, at 40–50 mm.

on the side of the cheek and on the fingers. He feels pricking normally on face and fingers.

The injury to the skull, caused by the fall in 1869, is indicated by an irregular depression existing on the vertex, within an inch to the right of the median line, in a plane passing vertically through the external auditory meatus.

The attacks of limited epilepsy were much reduced in frequency, but not interrupted for any length of time by the systematic use of the bromides pushed to the limit of prudence. The partial hemiplegia increased.

June 28th.—The weakness of the left leg has greatly increased; patient is unable to stand or walk without help; the fingers can be moved a little, and a degree of contraction has appeared in the elbow and hand.

Sept. 13th.—Patient had a slight general convulsion, with loss of consciousness, last night.

December 3d.—Is growing gradually worse. The ophthalmoscope shows fulness of veins, but no neuro-retinitis. Patient lies on his back, with left arm strongly flexed; there is some opisthotonus; complains of being deaf in right ear. Axillary temperature 36.4° C.

December 7th.—Complains of pain in right arm and leg, and in posterior part of head on the right side: pupils are equal and of medium size. There is no distortion of face, and the tongue comes out straight. The left upper extremity is completely paralyzed; the forearm and hand flexed and rigid. Left lower extremity is deprived of voluntary power, and lies rigidly extended. The neck is rigid, with tendency to opisthotonus. During the examination (and at other times) patient turns his head and eyes away from the palsied side—*déviation conjuguée.* Contact and pinching are felt on the palsied side. Pinching produces reflex spasm in the left limbs, of the nature of spinal epilepsy, *i. e.*, tonico-clonic spasm. The veins of the neck and forehead, especially on the right side, are unnaturally distended. An ophthalmoscopic examination shows no neuro-retinitis. Pulse 108, axillary temperature 37.1° C.

Localized and general convulsions recur from time to time. Patient is semi-conscious.

Death occurred on December 23, 1875.

The body is much emaciated, and rigor mortis is well marked, especially on the left side. On removing the scalp there occurs a

large escape of blood from enormously distended veins. The occipital veins on the left side are just perceptible, while on the right side they are 6 mm. in diameter. The frontal veins on both sides are much developed, but not filled with blood, because the body has lain upon its back. The right frontal bone is the seat of marked irregularities, and is rough and fissured between the median line and the external angular process. On the top of the skull, on a line passing vertically in front of the external auditory meatus, and 12.5 mm. from the median line, are two depressions about the size of peas. One of these contains a varicose vein, and a sound passed through the aperture seems to strike against the dura mater.

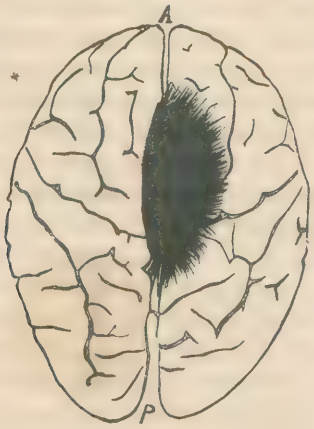
After sawing the skull through in the usual manner, the brain is divided horizontally without removing the skull-cap. The inferior half of the brain, examined as it lies in the fossæ of the skull, presents the following peculiarities: The right half of the brain is much enlarged, and the lateral ventricle and septum lucidum are forced over to the left. On the left side, the corpus striatum, thalamus, and convolutions appear healthy. On the right side the intra-ventricular nucleus of the corpus striatum alone is normal. Its extra-ventricular nucleus is undistinguishable. The white matter forward and outside of the corpus striatum, and in the neighborhood of the optic thalamus, is of a creamy consistency. The external and posterior half of the thalamus is involved in this softening. The convolutions directly outside of the thalamus are very much crowded together. At the base of the brain the convolutions lying immediately over the right olfactory bulb are very much softened, as are also the convolutions of the right sphenoidal lobe. The inferior convolutions of the occipital lobe appear normal. The white matter of the apex of the right anterior lobe is so soft that it is torn through in removal.

The upper half of the cerebrum, still lying in the skull, appears as follows: The septum lucidum is displaced to the left of the median line fully one-half inch. The white substance of the right hemisphere is the seat of a tumor, larger than a hen's egg, movable in the substance of the hemisphere, and of the consistence of glandular tissue. The opto-striate bodies of the right side are strongly compressed by the tumor, and so are the convolutions of the parietal region. The white matter in front and behind the growth is softened.

The upper half of the brain is carefully detached from the cal-

varium, by pushing the fingers between the dura mater and the bone. No difficulty is experienced in doing this until the neighborhood of the external depression in the bone is reached, when the dura mater is felt to be strongly adherent to the skull. With some difficulty the adhesion is broken up and the brain removed.

A careful examination of the convexity of the brain, thus exposed, shows the dura mater depressed and firmly adherent to the convolutions on the right side of the longitudinal fissure. There is some bony formation in the depressed adherent part of the dura. Around the patch, which is 25 mm. in diameter, the dura mater is moderately adherent to the anterior lobe over an oval space, measuring 75 mm. longitudinally, and 38 mm. transversely from the median line. The longitudinal fissure opposite this patch is obliterated by adhesions, and these are also present 13 mm. to the left of the median line.



CASE VI.—PATCH OF THICKENED DURA MATER ON TOP OF RIGHT HEMISPHERE.

Just to the right of the median line, opposite the depressed adherent spot in the dura, the skull presents a marked thickening and roughness. This tumor-like development of bone is 25 mm. anterior to the opening in the skull above described. A transverse section of the bone at this point shows it to be 13 mm. thick, and much condensed.

Transverse sections through the pons Varolii show no lesion except hyperæmia.

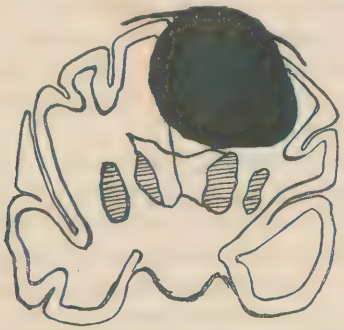
The fossæ of the skull are unusually deep, and irregularly furrowed and hilly.

The upper half of the left hemisphere, after hardening in bichromate of potassa, is examined by means of vertical transverse sections. The surfaces of these sections show that the tumor is very much larger than would appear from an inspection of the convexity of the hemisphere, or of the horizontal section above the ventricles. In fact, almost the whole of the anterior two-thirds of the upper half of the right brain is occupied by the growth, which has partly pushed aside and partly taken the place of the nervous tissue.

A transverse vertical section made through the anterior third of the thickened patch of dura mater reveals that the tumor occupies the whole thickness of the hemisphere above the opto-striate bodies, and that these bodies are strongly pressed upon. The mass of the tumor is so considerable as to press upon the inner surface of the left hemisphere.

A transverse vertical section through the middle of the thickened patch of dura mater and the cerebrum shows an appearance represented in Fig. 2. The tumor, continuous with the dura mater, extends deeply into the substance of the right hemisphere, down to the level of the roof of the lateral ventricle. Besides, this roof and the falx cerebri are much displaced by the growth.

Posterior to this level the tumor diminishes rapidly in size, though still pushing over to the left of the median line.



CASE VI.—TRANSVERSE VERTICAL SECTION THROUGH HEMISPHERES, SHOWING RELATIVE SIZE AND POSITION OF THE TUMOR.

A transverse vertical section made in the hinder part of the brain (beyond diseased dura mater) shows the tumor only as a small nodule in the upper inner part of the section-surface, lying wholly in the white substance.

The great longitudinal sinus is obliterated for a space of nearly 50 mm. in the thickest part of the patch of dura mater.

Microscopical examination showed the tumor to be an alveolar sarcoma in parts, and in others a common sarcoma.

As regards the connection between these lesions and the symptoms during life:

These symptoms were at first epilepsy, for nearly two years, followed by partial hemiplegia on the left side of the body, by incomplete localized epileptiform spasms in the left cheek, neck, and upper extremity, and the scene closed with complete left hemiplegia and a few general epileptic attacks.

It seems right to me to connect the above symptoms with the lesions in the following manner:

The first manifestations, general epileptic seizures, were caused by the development of thickening of the skull (internal plate), inflammation of the dura mater, and irritation of the cerebral substance.

As the pachymeningitis increased, with formation of bony spicules in its substance, the inflammation extended along the pia mater farther outward and backward, and thus reached parts of the convolutions which lie next to the fissure of Rolando and above the upper end of the fissure of Sylvius. These parts cover the regions numbered 2, 3, 4, and 6, in Ferrier's chart of the probable motor centres in the human brain, as deduced from experiments on apes.

By this irritation, there were caused the peculiar spasmodic movements of the muscles on the left side of the face and neck, and of the left hand and arm. Ferrier considers the regions numbered 2, 3, 4, and 6, as motor centres for the hand and arm chiefly.

There was also partial left hemiplegia. Later still, sarcoma was developed from the pachymeningitis; the malignant growth rapidly extended in all directions, substituting itself for the nervous tissue, and producing powerful compression-effects in all directions, but chiefly downward upon the opto-striate bodies.

In this terminal stage of the disease, the phenomena were complete left hemiplegia, a few general convulsions, no localized spasm.

As an additional sign of extensive lesion of the hemisphere, we may note the conjugate deviation of the eyes, away from the paralyzed side, and toward the injured hemisphere.

Why, with such an enormous tumor, and such an increase of intra-cranial pressure as must have existed, there was no

amblyopia, neuro-retinitis, atrophy of the optic nerves, diplopia or hemiopia, is a very puzzling question. This case is the second one of large cerebral tumor without choked disks or atrophy of the optic nerves which I observed in 1875.

In conclusion, I think that this case may be looked upon as corroborative of Hughling Jackson's, and Ferrier's theory of the existence of excitable motor districts, in some way connected with the motions of the face and forearm, in the upper median convolutions of the cerebrum—the ascending frontal and ascending parietal convolutions.

CASE VII.—*Pneumo-pyo-thorax; suppurative cerebral meningitis; abscesses in both hemispheres: localized epileptiform spasms in left hand, arm, and face; no paralysis or aphasia.*

For notes of the following remarkable case I am indebted to my friend, Dr. F. P. Kinnicutt.

The patient, a lad of thirteen, had been under the care of Drs. William H. Draper and Kinnicutt for empyema during several years.

During the last year of his life, the patient had a number of attacks of what was called slight septicæmia, characterized by moderate chill, fever, and diminished secretion from the pectoral fistula. The checking of the outflow of pus was held to be the cause of these attacks.

On February 18th, 1877, there occurred a severe chill, followed by fever, with a temperature of 37.7° and 38.8° C., lasting forty-eight hours. There was severe diffused headache. Dr. Kinnicutt was called and made the following observations:

On February 23d, patient felt very well, and it was thought that this attack had terminated favorably, like the others. But suddenly, while engaged in play, there occurred a twitching in the middle finger of the left hand; the spasm soon extended to the forearm. The entire spasm (clonic in form) only lasted a few seconds, and rather amused the boy. A few hours later a second attack took place, and was witnessed by Dr. Kinnicutt. Patient was aware of beginning of spasm by an almost imperceptible tremulousness of the facial muscles on the left side, followed, a few seconds later, by clonic spasm of the left middle finger and thumb (the former being strongly flexed, the latter adducted and flexed), succeeded by clonic flexions of the forearm, and evident clonic spasm of the left side of the face. The entire seizure lasted about 60 seconds, and during it there

was no spasm of any part but the left face, arm, forearm, and fingers. There was no loss of consciousness, and patient conversed intelligently during the attack. The pupils and vision remained normal. Not a trace of local or general paralysis followed the spasm (contrast to Hitzig's case). There was no anæsthesia or abnormal sensation (aura or numbness) in the affected parts before, during, or after the attack.

The headache, of which he has complained since the 18th, has grown much worse; it is not localized, but is more violent in the occipital region; the pain extending into the nape of the neck.

There were five or six of these hemiplegic spasms on the 23d.

I saw the patient in consultation with Dr. Kinnicutt that evening, and a careful examination failed to reveal any objective symptom except a buccal temperature of about 37.7° C. The pulse was proportionately rapid, but not irregular. Complaint was made of severe headache, as above. No trace of paralysis or disorder of sensibility; no nausea; mind remarkably clear; patient not anxious. Ophthalmoscope shows only somewhat enlarged retinal veins.

I agreed with Dr. Kinnicutt in diagnosticating a meningitis, probably of a tuberculous nature; and I advanced the view that there was a lesion of the convolutions on the right side, in the neighborhood of the fissure of Sylvius, involving the excitable district of the cortex, and producing the localized epilepsy.

February 24th.—At 6 o'clock A.M., had a general convulsion, followed by vomiting; T. 37.1°, P. 108 and regular. At 2.30 P.M., T. 38.8°, P. 100; headache violent; vomited once. At 10 P.M., T. 38.5°, P. 100 regular; R. 32 and regular.

February 25th.—At 10.30 A.M., T. 39°, P. 100 regular. At 5 P.M., T. 38.5°, P. 100 regular. At 10 P.M., T. 37.7°, P. 100. Vomited twice during afternoon; no new symptoms; headache violent; urine albuminous.

February 26th.—At 10 A.M., T. 39°. At 3.15 P.M., T. 37.7°, P. 94, slightly irregular. At 10.30 P.M., T. 37.5°, P. 88, slightly irregular. Vomited once in twenty-four hours.

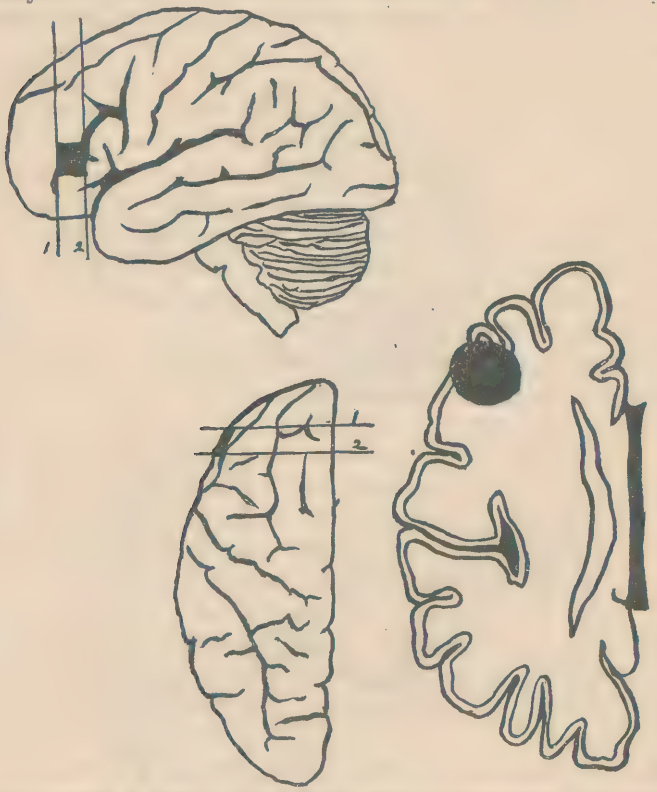
February 27th.—At 2.30 P.M., T. 37.5°, P. 80, R. 20. At 10 P.M., T. 38°, P. 88, irregular, R. 16–20, irregular.

February 28th.—At 5.30 A.M., pain in head intense; no motor disturbance of any kind; pupils normal and symmetrical; pulse and respiration irregular, the latter exhibiting the Cheyne-Stokes

phenomenon typically. The mind is perfectly clear; strength good.

At 6 A.M., there occurred a general convulsion, followed by several others in rapid succession. There was loss of consciousness, and death took place quietly at 7.40 A.M.

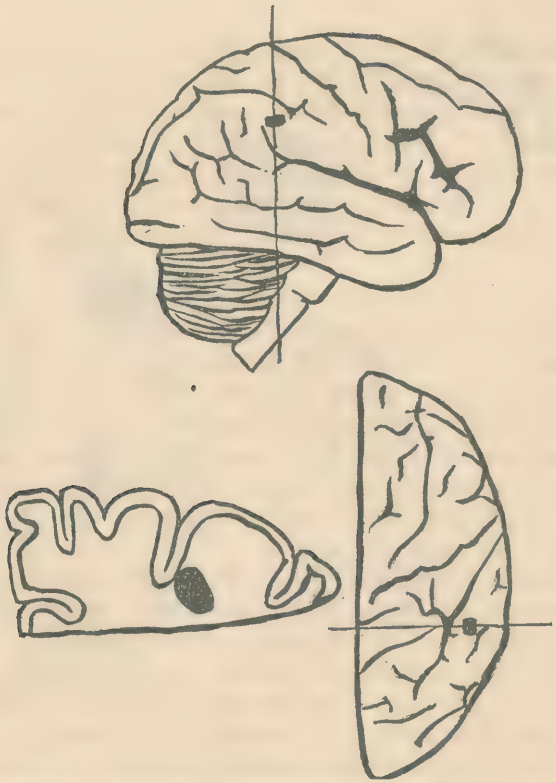
The treatment employed by Drs. Draper and Kinnicutt consisted in blisters to the neck, the giving of iodide of potassium, and, at last, to alleviate the severe cephalalgia, morphia hypodermically.



CASE VII.—ABSCESS IN LOWER PART OF SECOND FRONTAL CONVOLUTION, LEFT SIDE: NO SYMPTOMS.

I made the post-mortem examination at 3 o'clock P.M. on March 1st, in the presence of Drs. Draper and Kinnicutt. The condition of the left lung and remains of the pleura was one of great interest, but details on this point would here be out of place.

As regards the encephalon, we found a meningitis most marked at the base and sides of the brain, with purulent exudation in the meshes of the pia mater. There were no tubercles to be seen, and no marked lesion was to be found on the surface of the right hemisphere. On the left side, however, on the anterior lobe in front of the fissure of Sylvius, was a softened spot, probably an abscess. On carefully slicing the brain, a second lesion was found, however, and that in the right hemisphere just above the posterior extremity of the fissure of Sylvius. It is well worth while to state precisely the location and limits of these two localized lesions.



CASE VII.—ABSCESS IN RIGHT SECOND PARIETAL CONVOLUTION; CAUSE OF SPASM IN LEFT HAND AND FACE.

The first abscess, measuring 2 cent. in diameter, and lined by a soft membrane, nearly 1 mm. thick, lay in the lower part of the second frontal convolution, and the anterior border of the

third frontal convolution, on the left side, just in front of the speech-centre which has been referred to as injured in the cases of aphasia recorded in this essay. I am prepared to state, most positively, that the posterior part of the third convolution and island of Reil were perfectly healthy to the naked eye. The injured part is quite in front of Ferrier's centre, No. 9.

During life, no symptoms, motor, sensory, or intellectual, occurred which might be connected with this large irritating and destructive lesion. Consequently, it seems to be fair to conclude that in some human brains the lower part of the second frontal convolution is not excitable, and contains no motor cell-groups.

As regards the second abscess, not larger than a pea, it was found wholly in the white substance just beneath the cortex of the anterior part of the second parietal convolution on the right side, just above the upper extremity of the fissure of Sylvius. This lesion, which, in all probability, gave rise to the spasm in the left face and hand, is placed just behind the parts which Ferrier considers to be centres for motions of the hand and wrist.

If any conclusion is to be drawn from a study of the second lesion, it is that in man fibres for the face and hand pass farther downward and backward in the hemisphere than would be indicated by experiments on dogs and apes.

A fact certainly well worthy of remark is, that a small lesion in an excitable district of the brain may produce well-marked symptoms, whereas a much larger lesion may exist in non-excitable regions without giving any sign of its presence.

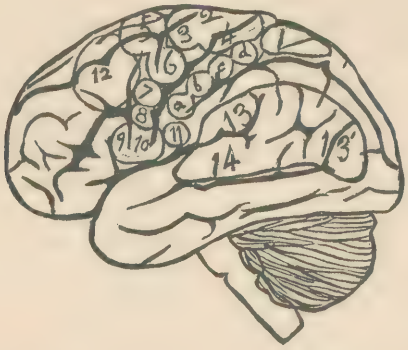
Conclusions: The three cases in Part I. fully bear out Broca's (and Ferrier's) hypothesis of the existence of a speech-centre in the posterior part of the left third frontal convolution, and in the anterior folds of the island of Reil.

The case in Part II. is unfortunately worth very little; still it may serve to show that a lesion of the ascending frontal convolution may cause paralysis of the arm (and leg) without affecting the lingual and facial muscles, or producing aphasia.

The two cases in Part III. appear to indicate that spasm, limited to one side of the face and one upper extremity, may be set up by irritation of quite distant parts of the cerebral cortex, extending from region No. 5 to below region d, in Ferrier's chart. In this

particular, these cases, while not contradicting experimental results, show that if there be such motor centres in man, they are not distributed quite as in monkeys and dogs.

The only case which appears to bear against the doctrine of localization of motor functions in the cerebral cortex is Case I. In this, it will be remembered, great recovery of voluntary power occurred, although a large part of the cortex of one hemisphere, including regions 6, 7, 11, a, b, and c, of Ferrier's chart, or, in other words, the so-called psycho-motor centres for the hand and forearm, the angle of the mouth and platysma, and those for the hand and wrist, were irreparably injured, nay, destroyed.



FERRIER'S CHART.—1, CENTRE FOR LEG; 2, 3, 4, FOR ARMS AND LEGS; 5, EXTENSION OF ARM AND HAND; 6, FOR BICEPS OF ARM; 7, 8, FOR ANGLE OF MOUTH; 9, 10, FOR LIPS AND TONGUE (BROCA'S CENTRE); 11, FOR PLATYSMA; 12, FOR HEAD, EYES, AND PUPILS; A, B, C, D (ASCENDING FRONTAL CONVOLUTION), FOR HAND AND WRIST; 13, 13′, 14, CENTRES FOR VISION AND HEARING.

I append a reproduction of Ferrier's chart of psycho-motor centres to render easy the determination of the injured parts in the above cases.

The problem of localization of functions in the cerebral cortex is a grand one, and well worthy of thorough study. I am not prepared to accept the hypothesis as enunciated by Ferrier, yet it seems to me in the highest degree rash and unscientific to call it baseless and worthless, because a few recent negative cases can be brought forward against it. As for old cases bearing on these questions, I wholly reject them as cracked or weak links in a chain that must be made only of approximately perfect joints. It is surely the duty of all medical men to publish cases negatively

or positively bearing on this question, but only when their observations have been made in an exact manner.

With a large accumulation of precise autopsies, with careful experiments by physiologists, with more embryological knowledge, and with unbiassed comparative anatomy, some one may, in a few years, study and solve the problem.